EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	2	"6599943".pn.	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:29
L2	0	tirhydroxypurine	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 17:40
L3	24	trihydroxypurine	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 17:40
L4	9	l3 and (ischemia or ischemic or heart failure)	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 17:40
L5	199	hydroxyguanidine	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 17:57
L6	10	l5 and (antitrypsin or antielastase or antiproteinase)	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 17:57
L7	1	"9823565"	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:09
L8	40	peroxynitrite adj scavengers	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:30
L9	40	peroxynitrite adj scavenger	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:57
L10	34	l9 and (ischemia or ischemic or myocardial or stroke or cerebrovascular)	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:58
L12	300	shapiro and antitrypsin	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:35
L13	20	l12 and antielastase	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:56
L14	1	peroxynirite adj scavenger	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:56
L15	47601	l9 or (scavenger)	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:57
L16	1000	l15 and ((uric adj acid) or dihydrorhodamine)	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:58

EAST Search History

L17	358	l16 and (ischemia or ischemic or myocardial or stroke or cerebrovascular)	US-PGPUB; USPAT; DERWENT	OR	ON	2008/01/03 18:59

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     (FILE 'HOME' ENTERED AT 17:09:46 ON 03 JAN 2008)

     FILE 'REGISTRY' ENTERED AT 17:10:01 ON 03 JAN 2008
L1             3 S DIHYDRORHODAMINE
L2             1 S TRIHYDROXYPURINE
L3           305 S RHODAMINE
L4             4 S L1 OR L2

     FILE 'CAPLUS' ENTERED AT 17:13:07 ON 03 JAN 2008
L5         17079 S L1 OR L2
L6           278 S L5 AND (ISCHEMIA REPERFUSION OR MYOCARDIAL INFARCTION OR HEAR
L7           143 S L6 AND PD <=2003
L8           143 FOCUS L7 1-
L9           143 S L8
L10           16 S L8 AND (COMBINATION OR COMB? OR COADMIN? OR CONCURRENT OR TOG
L11            0 S L6 AND (ANTITRYPSIN OR ANTIELASTASE OR ANTIPROTEINASE OR PROL
L12           19 S L5 AND (ANTITRYPSIN OR ANTIELASTASE OR ANTIPROTEINASE OR PROL
L13          517 S L1 OR DIHYDRORHODAMINE OR D 633 OR D-R 6G OR DIHYDRORHODAMINE
L14           20 S L13 AND (ISCHEMIA REPERFUSION OR MYOCARDIAL INFARCTION OR HEA
L15           20 FOCUS L14 1-
L16          323 S L2 AND (ISCHEMIA REPERFUSION INJURY OR MYOCARDIAL INFARACTION
L17          195 S L16 AND PD <=2003
L18          195 FOCUS L17 1-

=>
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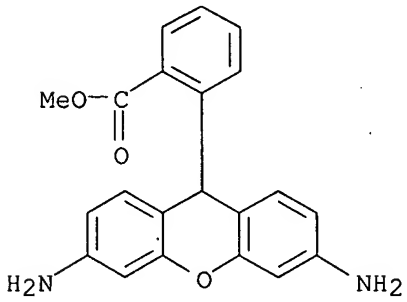
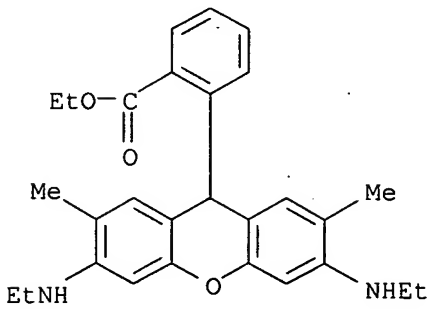
L1 ANSWER 1 OF 3 REGISTRY COPYRIGHT 2008 ACS on STN
RN 691855-47-7 REGISTRY
ED Entered STN: 11 Jun 2004
CN Benzoic acid, 2-(3,6-diamino-9H-xanthen-9-yl)-, methyl ester, dihydrochloride (9CI) (CA INDEX NAME)
OTHER NAMES:
CN **Dihydrorhodamine 123 dihydrochloride**
MF C21 H18 N2 O3 . 2 Cl H
SR CA
LC STN Files: CA, CAPLUS, USPATFULL
CRN (109244-58-8)



● 2 HCl

1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

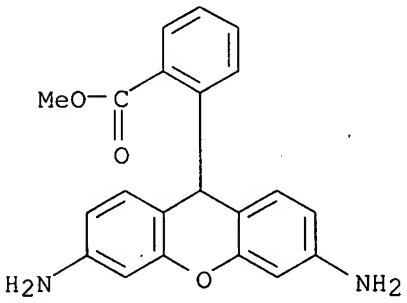
L1 ANSWER 2 OF 3 REGISTRY COPYRIGHT 2008 ACS on STN
RN 217176-83-5 REGISTRY
ED Entered STN: 15 Jan 1999
CN Benzoic acid, 2-[3,6-bis(ethylamino)-2,7-dimethyl-9H-xanthen-9-yl]-, ethyl ester (CA INDEX NAME)
OTHER NAMES:
CN D 633
CN d-R 6G
CN **Dihydrorhodamine 6G**
DR 470671-59-1
MF C28 H32 N2 O3
SR CAS Client Services
LC STN Files: CA, CAPLUS, CHEMCATS, TOXCENTER, USPATFULL



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

13 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
13 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L1 ANSWER 3 OF 3 REGISTRY COPYRIGHT 2008 ACS on STN
RN 109244-58-8 REGISTRY
ED Entered STN: 18 Jul 1987
CN Benzoic acid, 2-(3,6-diamino-9H-xanthen-9-yl)-, methyl ester (CA INDEX NAME)
OTHER NAMES:
CN D 23806
CN D 632
CN **Dihydrorhodamine 123**
MF C21 H18 N2 O3
CI COM
SR CA
LC STN Files: ADISNEWS, AGRICOLA, BEILSTEIN*, BIOSIS, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, EMBASE, MEDLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)



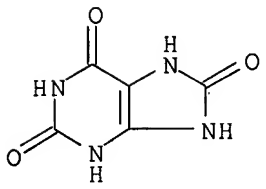
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

101 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
101 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> d l2

L2 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2008 ACS on STN
RN 69-93-2 REGISTRY
ED Entered STN: 16 Nov 1984
CN 1H-Purine-2,6,8(3H)-trione, 7,9-dihydro- (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Uric acid (8CI)
OTHER NAMES:
CN 1H-Purine-2,6,8-triol
CN **2,6,8-Trihydroxypurine**
CN 2,6,8-Trioxopurine
CN 2,6,8-Trioxypurine
CN Lithic acid
CN NSC 3975
CN Purine-2,6,8(1H,3H,9H)-trione
DR 13154-20-6, 530-13-2, 33278-42-1, 34318-07-5, 42911-25-1, 42911-27-3, 42911-28-4
MF C5 H4 N4 O3
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO,

CA, CABA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CIN, CSCHEM,
CSNB, DDFU, DETHERM*, DRUGU, EMBASE, GMELIN*, IFICDB, IFIPAT, IFIUDB,
IMSRESEARCH, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, PIRA, PROMT,
RTECS*, SPECINFO, TOXCENTER, ULIDAT, USPAT2, USPATFULL, USPATOLD
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

16899 REFERENCES IN FILE CA (1907 TO DATE)
140 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
16968 REFERENCES IN FILE CAPLUS (1907 TO DATE)
6 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=>

L3 ANSWER 305 OF 305 REGISTRY COPYRIGHT 2008 ACS on STN
RN 81-88-9 REGISTRY
ED Entered STN: 16 Nov 1984
CN Xanthylium, 9-(2-carboxyphenyl)-3,6-bis(diethylamino)-, chloride (1:1) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ammonium, [9-(o-carboxyphenyl)-6-(diethylamino)-3H-xanthen-3-ylidene]diethyl-, chloride (8CI)
OTHER NAMES:
CN 11411 Red
CN **ADC Rhodamine B**
CN **Aizen Rhodamine B**
CN **Aizen Rhodamine BH**
CN **Aizen Rhodamine BHC**
CN **Akiriku Rhodamine B**
CN Basazol Red 71P
CN Basic Rose Extract
CN Basic Rose Red
CN Basic Violet 10
CN Basonyl Red 540
CN Basonyl Red 545
CN Basonyl Red 545FL
CN Brilliant Pink B
CN C.I. 45170
CN C.I. Basic Violet 10
CN C.I. Food Red 15
CN Calcozine Red BX
CN **Calcozine Rhodamine BXP**
CN Cerise Toner X 1127
CN D and C Red No. 19
CN D&C Red 19
CN D&C Red No. 19
CN **Diabasic Rhodamine B**
CN Edicol Supra Rose B
CN Edicol Supra Rose BS
CN **Eriosin Rhodamine B**
CN FD And C Red No. 19
CN Flexo Red 540
CN **Hexacol Rhodamine B Extra**
CN **Ikada Rhodamine B**
CN Japan Red 213
CN Japan Red No. 213
CN LC 6100
CN **Mitsui Rhodamine BX**
CN OP 312
CN Red No. 213
CN Rheonine B
CN **Rhodamine 610 chloride**
CN **Rhodamine B**
CN **Rhodamine B 500**
CN **Rhodamine B 500 hydrochloride**
CN **Rhodamine B Extra**
CN **Rhodamine B Extra M 310**
CN **Rhodamine B Extra S**
CN **Rhodamine BA**
CN **Rhodamine BA Export**
CN **Rhodamine BN**
CN **Rhodamine BS**
CN **Rhodamine BX**
CN **Rhodamine BXL**
CN **Rhodamine BXP**
CN **Rhodamine FB**
CN **Rhodamine Lake Red B**

CN **Rhodamine O**
CN **Rhodamine S**
CN **Rhodamine S (Russian)**
CN **Rhodamine, tetraethyl-**
CN **Symulex Rhodamine B Toner F**
CN **Takaoka Rhodamine B**
CN **Tetraethylrhodamine**
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
DR 850856-47-2, 859039-47-7, 956491-27-3, 875572-56-8, 918962-66-0, 925914-34-7, 433215-26-0, 11111-29-8, 53664-59-8, 3521-79-7, 105480-59-9, 69319-23-9, 86513-49-7, 86893-15-4, 248928-56-5, 408346-58-7, 412909-17-2, 539821-35-7
MF C28 H31 N2 O3 . Cl
CI COM
LC STN Files: AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DRUGU, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, PIRA, PROMT, RTECS*, SPECINFO, TOXCENTER, USPAT2, USPATFULL, USPATOLD, VETU
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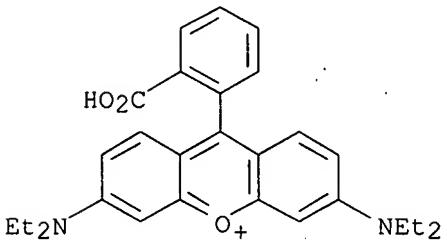
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (64381-98-2)



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PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

6758 REFERENCES IN FILE CA (1907 TO DATE)
435 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
6785 REFERENCES IN FILE CAPLUS (1907 TO DATE)
8 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

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L15 ANSWER 2 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1999:768521 CAPLUS
DOCUMENT NUMBER: 132:44938
TITLE: Enhanced ADP-ribosylation and its diminution by lipoamide after **ischemia-reperfusion** in perfused rat heart
AUTHOR(S): Szabados, Eszter; Fischer, Gabor M.; Gallyas, Ferenc., Jr.; Kispal, Gyula; Sumegi, Balazs
CORPORATE SOURCE: Department of Biochemistry, University Medical School Pecs, Pecs, 7624, Hung.
SOURCE: Free Radical Biology & Medicine (1999), 27(9/10), 1103-1113
CODEN: FRBMEH; ISSN: 0891-5849
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

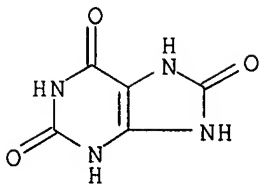
AB Poly-ADP-ribose polymerase (PARP) is considered to play an important role in oxidative cell damage. We assumed that **ischemia-reperfusion** resulting from the increasing reactive oxygen species (ROS) can lead to the activation of endogenous mono- and poly-ADP-ribosylation reactions and that the reduction of ROS level by lipoamide, a less known antioxidant, can reverse these unfavorable processes. Expts. were performed on isolated Langendorff hearts subjected to 60-min ischemia followed by reperfusion. ROS, malondialdehyde, DNA breaks, and NAD+ content were assayed in the hearts, and the ADP-ribosylation of cytoplasmic and nuclear proteins were determined by Western blot assay. **Ischemia-reperfusion** caused a moderate (30.2 ± 8%) increase in ROS production determined by the **dihydrorhodamine 123** method and significantly increased the malondialdehyde production (from <1 to 23 ± 2.7 nmol/mL), DNA damage (undamaged DNA decreased from 71 ± 7% to 23.1 ± 5%), and NAD+ catabolism. In addition, **ischemia-reperfusion** activated the mono-ADP-ribosylation of GRP78 and the self-ADP-ribosylation of the nuclear PARP. The perfusion of hearts with lipoamide significantly decreased the **ischemia-reperfusion**-induced cell membrane damage determined by enzyme release (LDH, CK, and GOT), decreased the ROS production, reduced the malondialdehyde production to 5.5 ± 2.4 nmol/mL, abolished DNA damage, and reduced NAD+ catabolism. The **ischemia-reperfusion**-induced activation of poly- and mono-ADP-ribosylation reactions were also reverted by lipoamide. In isolated rat heart mitochondria, dihydrolipoamide was found to be a better antioxidant than dihydrolipoic acid. **Ischemia-reperfusion** by ROS overprodn. and increasing DNA breaks activates PARP leading to accelerated NAD+ catabolism, impaired energy metabolism, and cell damage. Lipoamide by reducing ROS levels halts PARP activation and membrane damage and improves the recovery of postischemic myocardium.

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN T

L15 ANSWER 1 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1995:847181 CAPLUS
DOCUMENT NUMBER: 123:253251
TITLE: Peroxynitrite-mediated oxidation of **dihydrorhodamine 123** occurs in early stages of endotoxic and hemorrhagic shock and **ischemia-reperfusion** injury
AUTHOR(S): Szabo, Csaba; Salzman, Andrew L.; Ischiropoulos, Harry
CORPORATE SOURCE: Children's Hospital Medical Center, Division of Critical Care, 3333 Burnet Avenue, Cincinnati, OH, 45229, USA
SOURCE: FEBS Letters (1995), 372(2,3), 229-32
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To quantify peroxynitrite production during shock, the authors measured oxidation of **dihydrorhodamine 123** in rats. In endotoxic and hemorrhagic shock and splanchnic **ischemia-reperfusion**, **dihydrorhodamine** oxidation rapidly increased, which was prevented by inhibition of endothelial nitric oxide (NO) synthase (ecNOS). Thus, peroxynitrite is already formed at early stages of shock from ecNOS-derived NO. Overprodn. of NO by the inducible NOS at late shock was not associated with addnl. increases in **dihydrorhodamine** oxidation ecNOS inhibition enhanced **dihydrorhodamine** oxidation in control rats. These latter findings may be explained by NO-mediated inhibition of peroxynitrite-induced **dihydrorhodamine** oxidation, a phenomenon also observed in vitro.

L15 ANSWER 9 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:285166 CAPLUS
DOCUMENT NUMBER: 129:80401
TITLE: Complement activation following reoxygenation of hypoxic human endothelial cells: Role of intracellular reactive oxygen species, NF-κB and new protein synthesis
AUTHOR(S): Collard, Charles D.; Agah, Azin; Stahl, Gregory L.
CORPORATE SOURCE: Brigham and Women's Hospital, Department of Anesthesia, Center for Experimental Therapeutics and Reperfusion Injury, Harvard Medical School, Boston, MA, 02115, USA
SOURCE: Immunopharmacology (1998), 39(1), 39-50
CODEN: IMMUDP; ISSN: 0162-3109
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Complement plays an important role in **ischemia-reperfusion** injury. We recently demonstrated that reoxygenation of hypoxic human umbilical vein endothelial cells (HUVECs) activated the classical complement pathway and augmented iC3b deposition. In the present study, we investigated the potential role of oxygen-derived free radicals, NF-κB and new protein synthesis in this model. HUVECs subjected to 12 or 24 h hypoxic stress (1% O2) and then reoxygenated (0.5, 1, 2 or 3 h; 21% O2) in 30% human serum activated complement and deposited iC3b. Addition of hydrogen peroxide (H2O2; 1-100 μmol/l) to normoxic HUVECs increased iC3b deposition in a concentration-dependent manner. H2O2 (10 μmol/l), a concentration that did not significantly increase iC3b deposition on normoxic HUVECs, augmented iC3b deposition on hypoxic/reoxygenated HUVECs. We observed a significant increase in intracellular H2O2 and hydroxyl radical (OH·) production in hypoxic/reoxygenated HUVECs using **dihydrorhodamine 123**. Further, treatment of HUVECs with dimethylthiourea (DMTU, 1-100 μmol/l), deferoxamine (DEF, 1-100 μmol/l), or oxypurinol (10 μmol/l), but not superoxide dismutase (SOD, 500 U/mL), catalase (300 U/mL) or iron-loaded DEF, attenuated iC3b deposition following hypoxia/reoxygenation in a concentration-dependent manner. Western anal. demonstrated hypoxia-induced nuclear NF-κB translocation that increased with reoxygenation. Inhibition of new protein synthesis (i.e. cycloheximide) or inhibition of NF-κB (ALLN or SN-50) also significantly decreased iC3b deposition on hypoxic/reoxygenated HUVECs. We conclude that (1) hypoxic/reoxygenated HUVECs generate H2O2 and OH·; (2) treatment of HUVECs with cell permeable reactive oxygen species inhibitors/scavengers (i.e., DEF, DMTU, oxypurinol) but not large mol. weight inhibitors (i.e. catalase or SOD) significantly reduces iC3b deposition; and (3) inhibition of new protein synthesis or NF-κB activation attenuates iC3b deposition. These data suggest that iC3b deposition on the vascular endothelium may be regulated by intracellular oxygen-derived free radical induced activation of NF-κB, new protein synthesis and activation of the classical complement pathway during **ischemia/reperfusion**.
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE R

L18 ANSWER 10 OF 195 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:388241 CAPLUS
DOCUMENT NUMBER: 135:342454
TITLE: Uric acid in cachectic and noncachectic patients with chronic **heart failure**: relationship to leg vascular resistance
AUTHOR(S): Doehner, Wolfram; Rauchhaus, Mathias; Florea, Viorel G.; Sharma, Rakesh; Bolger, Aidan P.; Davos, Constantinos H.; Coats, Andrew J. S.; Anker, Stefan D.
CORPORATE SOURCE: Clinical Cardiology, National Heart and Lung Institute, Imperial College School of Medicine, London, SW3 6LY, UK
SOURCE: American Heart Journal (**2001**), 141(5), 792-799
CODEN: AHJOA2; ISSN: 0002-8703
PUBLISHER: Mosby, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Background Chronic **heart failure** (CHF) is a hyperuricemic state, and capillary endothelium is the predominant site of xanthine oxidase in the vasculature. Upregulated xanthine oxidase activity (through production of toxic free radicals) may contribute to impaired regulation of vascular tone in CHF. We aimed to study the relationship between serum uric acid levels and leg vascular resistance in patients with CHF with and without cachexia and in healthy control subjects. Methods In 23 cachectic and 44 noncachectic patients with CHF (age, 62 ± 1 yr, mean ± SEM) and 10 healthy control subjects (age, 68 ± 1 yr), we assessed leg resting and postischemic peak vascular resistance (calculated from mean blood pressure and leg blood flow by venous occlusion plethysmog.). Results Cachectic patients, compared with noncachectic patients and control subjects, had the highest uric acid levels (612 ± 36 vs 459 ± 18 and 346 ± 21 μmol/L, resp., both $P < .0001$) and the lowest peak leg blood flow and vascular reactivity (reduction of leg vascular resistance from resting to postischemic conditions: 83% vs 88% and 90%, both $P < .005$). In all patients, postischemic vascular resistance correlated significantly and independently of age with uric acid ($r = 0.61$), creatinine ($r = 0.47$, both $P < .0001$), peakVO2 ($r = 0.34$), and New York Heart Association class ($r = 0.33$, both $P < .01$). This correlation was not present in healthy control subjects ($r = -0.04$, $P = .9$). In multivariate and stepwise regression analyses, serum uric acid emerged as the strongest predictor of peak leg vascular resistance (standardized coefficient = 0.61, $P < .0001$) independent of age, peakVO2, creatinine, New York Heart Association class, and diuretic dose. Conclusions Hyperuricemia and postischemic leg vascular resistance are highest in cachectic patients with CHF, and both are directly related independent of diuretic dose and kidney function. The xanthine oxidase metabolic pathway may contribute to impaired vasodilator capacity in CHF.
IT **69-93-2**, Uric acid, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(uric acid in human cachectic and noncachectic patients with chronic **heart failure** in relationship to leg vascular resistance)
RN 69-93-2 CAPLUS
CN 1H-Purine-2,6,8(3H)-trione, 7,9-dihydro- (CA INDEX NAME)



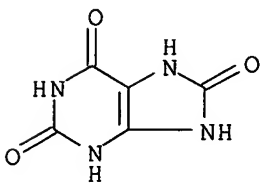
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 1 OF 195 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1999:21396 CAPLUS
DOCUMENT NUMBER: 130:221418
TITLE: Uric acid in chronic **heart failure** : a marker of chronic inflammation
AUTHOR(S): Leyva, F.; Anker, S. D.; Godsland, I. F.; Teixeira, M.; Hellewell, P. G.; Kox, W. J.; Poole-Wilson, P. A.; Coats, A. J. S.
CORPORATE SOURCE: Department of Cardiac Medicine, National Heart and Lung Institute, Imperial College School of Medicine, London, SW3 6LY, UK
SOURCE: European Heart Journal (**1998**), 19(12), 1814-1822
CODEN: EHJODF; ISSN: 0195-668X
PUBLISHER: W. B. Saunders Co. Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Chronic **heart failure** is associated with hyperuricemia and elevations in circulating markers of inflammation. Activation of xanthine oxidase, through free radical release, causes leukocyte and endothelial cell activation. Assocns. could therefore be expected between serum uric acid level, as a marker of increased xanthine oxidase activity, and markers of inflammation. We have explored these assocns. in patients with chronic **heart failure**, taking into account the hyperuricemic effects of diuretic therapy and insulin resistance. Circulating uric acid and markers of inflammation were measured in 39 male patients with chronic **heart failure** and 16 healthy controls. All patients underwent a metabolic assessment, which provided a measure of insulin sensitivity (i.v. glucose tolerance tests and minimal modeling anal.). Compared to controls, patients with chronic **heart failure** had significantly higher levels of circulating uric acid, interleukin-6, soluble tumor necrosis factor receptor (sTNFR)-1, soluble intercellular adhesion mol.-1 (ICAM-1, all $P<0.001$), E-selectin and sTNFR2 (both $P<0.05$). In patients with chronic **heart failure**, serum uric acid concns. correlated with circulating levels of sTNFR1 ($r=0.74$), interleukin-6 ($r=0.66$), sTNFR2 ($r=0.63$), TNFα ($r=0.60$) (all $P<0.001$), and ICAM-1 ($r=0.41$, $P<0.01$). In stepwise regression analyses, serum uric acid emerged as the strongest predictor of ICAM-1, interleukin-6, TNF, sTNFR1 and sTNFR2, independent of diuretic dose, age, body mass index, alc. intake, serum creatinine, plasma insulin and glucose, and insulin sensitivity. Serum uric acid is strongly related to circulating markers of inflammation in patients with chronic **heart failure**. This is consistent with a role for increased xanthine oxidase activity in the inflammatory response in patients with chronic **heart failure**.

IT **69-93-2**, Uric acid, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(uric acid in human serum is strongly related to circulating markers of inflammation in patients with chronic **heart failure**)

RN 69-93-2 CAPLUS
CN 1H-Purine-2,6,8(3H)-trione, 7,9-dihydro- (CA INDEX NAME)

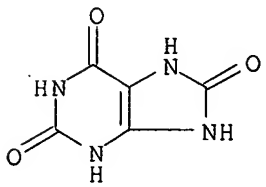


REFERENCE COUNT: 71 THERE ARE 71 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 2 OF 195 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:575572 CAPLUS
DOCUMENT NUMBER: 136:64038
TITLE: **Ischemia/reperfusion injury** of rat small intestine: the effect of allopurinol dosage
AUTHOR(S): Ciz, M.; Cizova, H.; Lojek, A.; Kubala, L.; Papezikova, I.
CORPORATE SOURCE: Institute of Biophysics, Academy of Sciences of the Czech Republic, Brno, Czech Rep.
SOURCE: Transplantation Proceedings (**2001**), 33(5), 2871-2873
CODEN: TRPPA8; ISSN: 0041-1345
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of allopurinol on the elimination of xanthine oxidase-derived free radicals in rats with intestinal ischemia/reperfusion (I/R) were studied. Three exptl. rat groups were studied: (a) without allopurinol, (b) allopurinol in drinking water for a week prior to surgery, and (c) allopurinol i.p. The protective effects of allopurinol in the ischemia/reperfusion model of rat small intestine were observed only when drug was given i.p. Since the major protective effects of allopurinol were seen in the decreased number and activity of neutrophils, it can be speculated that XO-derived reactive oxygen species do not contribute directly to the development of oxidative injury during I/R. The xanthine/xanthine oxidase system was more likely responsible for the induction of addnl. damage caused by other systems such as mobilized and activated neutrophils.

IT **69-93-2**, Uric acid, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(effect of allopurinol on small intestine **ischemia/reperfusion injury**: mechanism of protective effect)
RN 69-93-2 CAPLUS
CN 1H-Purine-2,6,8(3H)-trione, 7,9-dihydro- (CA INDEX NAME)



REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

Inventor Information for 10/669251

Inventor Name	City	State/Country
SHAPIRO, LELAND	DENVER	COLORADO

Appln Info | Contents | Petition Info | Atty/Agent Info | Continuity/Reexam | Foreign

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